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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/006,856	12/06/2001	Kevin` P. Baker	GNE:2830P1C14	8365

9157 7590 01/24/2008

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EXAMINER
VOGEL, NANCY S

ART UNIT	PAPER NUMBER
1636	

MAIL DATE	DELIVERY MODE
01/24/2008	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/006,856	BAKER ET AL.	
	Examiner	Art Unit	
	Nancy T. Vogel	1636	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *04 October 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *28-35 and 38-47* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *28-35 and 38-47* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☒ Other: *sequence alignment*.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 10/4/07 has been entered.

The rejection of claims 28-35, 38-40 are no longer included in the rejection under 35 USC 101 and associated 112 first paragraph, since the utility for the polypeptide, i.e. as a stimulator of uptake of glucose or FFA in adipocytes, is accepted.

Claim Rejections - 35 USC § 101

Claims 41-47 are rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a credible, specific and substantial asserted utility or a well established utility.

Claims 41-47 are also rejected under 35 U.S.C. 112, first paragraph. Specifically, since the claimed invention is not supported by either a credible, specific and substantial asserted utility or a well established utility for the reasons set forth above, one skilled in the art clearly would not know how to use the claimed invention.

This is a new rejection, based upon several new references which are cited for support, and based upon new reasoning. Specifically, the

examiner no longer asserts that mRNA levels are not predictive of polypeptide levels. Therefore, the following references are no longer being relied upon to support the rejections: Chen et al., Hu et al., Haynes et al., Lian et al., Fessler et al., Nagaraja et al., Waghray et al., Sagnaliev et al., Lilley et al., Wildsmith et al., King et al., Celis et al., and Madoz-Gurpide et al. The following references cited and discussed by Applicant pertaining to the mRNA/polypeptide correlation issue will no longer be addressed: Futcher et al., Alberts and Lewin, Meric et al., Zhigang et al., Wang et al., Munaut et al. The basis of the maintained rejections is solely that gene amplification levels are not predictive of mRNA or polypeptide levels.

The claims are directed to the polypeptide of SEQ ID NO: 194 and variants thereof. The specification discloses the polypeptide of SEQ ID NO: 194, also known as PRO1303. The claims recite that the polypeptides are encoded by nucleic acid that is amplified in lung and colon tumors. In the specification, a gene amplification assay in which genomic DNA encoding PRO1303 had a Ct value of at least 1.0 for

At pages 539-555 of the specification, Example 170 discloses a gene amplification assay in which genomic DNA encoding PRO1112 had a ACt value of at least 1.0 for 5 lung or colon tumor samples when compared to a pooled control of blood DNA from several healthy volunteers. Example 143 asserts that gene amplification is associated with overexpression of the gene product (i.e., the polypeptide), indicating that the polypeptides are useful targets for therapeutic intervention in cancer and diagnostic

determination of the presence of cancer . A delta Ct is defined as the threshold PCR cycle, or the cycle at which the reporter signal accumulates above the background level of fluorescence. The specification further indicates that delta Ct is used as a quantitative measurement of the relative number of starting copies of a particular target sequence in a nucleic acid sample when comparing cancer DNA results to normal human DNA results. It is noted that at page 507, it is stated that samples are used if their values are within 1 Ct of the 'normal standard'. It is further noted that the ACt values are expressed with values to one one-hundredth of a unit (e.g. 1.29).

First, there are several problems with the data provided in this example. The art recognizes that lung and colon epithelium is can be aneuploid without the presence of cancer. Specifically, Hittelman (2001, Ann. N. Y. Acad. Sci. 952:1-12) reports that lung epithelium is at risk for cellular damage due to direct exposure to environmental pollutants and carcinogens, which result in aneuploidy before the epithelial cells turn cancerous. See especially p. 4, Figure 4. The gene amplification assay in the instant specification does not provide a comparison between the lung tumor samples and normal lung epithelium and does not correct for aneuploidy. Thus it is not clear that PRO1112 is amplified in cancerous lung epithelium more than in damaged (non\-cancerous) lung epithelium. One skilled in the art would not conclude that PRO1303 is a diagnostic probe for lung cancer unless it is clear that PRO1303 is amplified to a clearly greater extent in true lung tumor tissue relative to non-cancerous lung epithelium. Regarding colon tissue, pre-malignant lesions and ulcerative colitis have been associated with aneuploidy. See Fleischhacker et al. (1995, Modern Pathology

8:360-365), especially p. 360, 1st paragraph of introduction. The gene amplification assay in the instant specification does not provide a comparison between the colon tumor samples and normal colon epithelium and does not correct for aneuploidy. Thus it is not clear that PRO1303 is amplified in cancerous colon epithelium more than in damaged (non-cancerous) colon epithelium. One skilled in the art would not conclude that PRO1303 is a diagnostic probe for colon cancer unless it is clear that PRO1303 is amplified to a clearly greater extent in true colon tumor tissue relative to non-cancerous colon epithelium.

Second, even if the data had been corrected for aneuploidy and a proper control had been used, the data have no bearing on the utility of the claimed PRO1303 polypeptide. In order for PRO1303 polypeptide to be overexpressed in tumors, amplified genomic DNA would have to correlate with increased mRNA levels and increased polypeptide levels. No data regarding PRO1303 mRNA or PRO1303 polypeptide levels in lung or colon tumors have been brought forth on the record. The art discloses that a correlation between genomic DNA levels and mRNA levels cannot be presumed, nor can any correlation between genomic DNA levels and polypeptide levels. A specific example of the lack of correlation between genomic DNA amplification

and increased mRNA expression is provided by Pennica et al. (1998, PNAS USA 95:14717-14722), who disclose that: "An analysis of WISP-1 gene amplification and expression in human colon tumors showed a correlation between DNA amplification and overexpression, whereas overexpression of WISP-3 RNA was seen in the absence of DNA amplification. In contrast, WISP-2 DNA was amplified in the colon tumors, but its

mRNA expression was significantly reduced in the majority of tumors compared with the expression in normal colonic mucosa from the same patient.'

See p. 14722, second paragraph of left column; pp. 14720-14721, "Amplification and Aberrant Expression of WISPs in Human Colon Tumors." Another specific example is provided by Konopka et al. (Proc. Natl. Acad. Sci. (1986) 83:4049-4052), who state that "Protein expression is not related to amplification of the abl gene but to variation in the level of bcr-abl mRNA produced from a single Phl template" (see abstract).

The general concept of gene amplification's lack of correlation with mRNA/protein overexpression in cancer tissue is addressed by Sen (2000, Curr. Opin. Oncol. 12:82-88). Specifically, Sen teaches that cancerous tissue is known to be aneuploid, that is, having an abnormal number of chromosomes. A slight.amplification of a gene does not necessarily correlate with overexpression in a cancer tissue, but can merely be an indication that the cancer tissue is aneuploid. Hittelman (2001, Ann. N. Y. Acad. Sci. 952:1-12) also speaks to this issue. Again, the data in the specification were. not corrected for such aneuploidy events. Furthermore, Godbout et al. (1998, J. Biol. Chem. 273(33):21161-8) teach a general lack of correlation between gene amplification and mRNA/protein overexpression. The abstract of Godbout teaches "The DEAD box gene, DDXI, is a putative RNA helicase that is co-amplified with MYCN in a subset of retinoblastoma (RB) and neuroblastoma (NB) tumors and cell lines. Although gene amplification usually involves hundreds to thousands of kilobase pairs of DNA, a number of studies suggest that co-amplified genes are only overexpressed if they provide a selective advantage to the cells in which they are amplified." (emphasis

added). The protein encoded by the DDX gone had been characterized as being a putative RNA helicase, a type of enzyme that would be expected to confer a selective advantage to the cells in which it (the DDX gone) was amplified. On page 21167, right column, first full paragraph, Godbout et al. state "It is generally accepted that co-amplified genes are not over-expressed unless they provide a selective growth advantage to the cell (48, 49). For example, although ERBA is closely linked to ERBB2 in breast cancer and both genes are commonly amplified in these tumors, ERBA is not overexpressed (48). Similarly, three genes mapping to 12q13-14 (CDK4, SAS and MDM2) are overexpressed in a high percentage of malignant gliomas showing amplification of this chromosomal region, while other genes mapping to this region (GADD153, GL1, and A2MR) are rarely overexpressed in gone-amplified malignant gliomas (50, 51). The first three genes are probably the main targets of the amplification process, while the latter three genes are probably incidentally included in the amplicons." (emphasis added). There is no evidence in the instant application that PRO1303 confers any growth advantage to a cell, and thus it cannot be presumed that the protein is overexpressed because the genomic DNA including the gone being studied gone is amplified.

An additional reference that provides evidence that gone amplification does not generally lead to increased transcript is Li et al. (2006; Oncogene, Vol. 25, pages 2628-2635). Li et al. used a functional approach that integrated simultaneous genomic and transcript microarray, proteomics, and tissue microarray analyses to directly identify putative oncogenes in lung adenocarcinoma. On page 2633, right column, Li et al. state:

"In our study, 68.8% of the genes showing over-representation in the genome did not show elevated transcript levels, implying that at least some of these genes are 'passenger' genes that are concurrently amplified because of their location with respect to amplicons but lack biological relevance in terms of the development of lung adenocarcinoma." Since more than half of the amplified genes were not overexpressed, Li et al. constitutes strong evidence that it is more likely than not that gene amplification does NOT correlate with increased protein levels, absent evidence that the protein has biological relevance in cancer. There is no such evidence for PRO1303.. The data do not support the specification's assertion that PRO1303 polypeptides and their antibodies can be used as cancer diagnostic agents. Significant further research would have been required of the skilled artisan to reasonably confirm that the PRO1303 polypeptide is overexpressed in any cancer to the extent that the polypeptide or antibodies that bind it could be used as cancer diagnostic agents, and thus the asserted utility is not substantial. In the absence of information regarding whether or not PRO1112 polypeptide levels are also different between specific cancerous and normal tissues, the proposed use of the PRO1303 polypeptides as diagnostic markers and therapeutic targets are simply starting points for further research and investigation into potential practical uses of the polypeptides and antibodies. See Brenner v. Manson, 148 U.S.P.Q. 689 (Sup. Ct., 1966), wherein the court held that:

"The basic quid pro quo contemplated by the Constitution and the Congress for granting a patent monopoly is the benefit derived by the public from an invention with substantial utility", "[u]nless and until a process is refined and developed to this point-where specific

benefit exists in currently available form-there is insufficient justification for permitting an applicant to engross what may prove to be a broad field", and "a patent is not a hunting license", "[i]t is not a reward for the search, but compensation for its successful conclusion."

In view of the preponderance of evidence supporting the rejections (Pennica et al., Konopka et al., Fleischhacker et al., Sen, Hittelman, Godbout et al., and Li et al)., the rejections are properly maintained.

The Declaration of Dr. Randy Scott, and Dr. Paul Polakis under 37 CFR 1.132 filed 10/4/07 is insufficient to overcome the rejection of claims 41-47 based upon 35 USC 101/112 first paragraph as set forth in the last Office action because: the current rejection is based upon the gene amplification as a utility. It is not longer being maintained that mRNA levels are not predictive of polypeptide levels.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 28-32, 39-47 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to

which it pertains, or with which it is most nearly connected, to make and/or use the invention.

This rejection is maintained for reasons of record.

Applicants have argued that the claims do not recite any variant, but only those in which the nucleic acid encoding the polypeptide is amplified in lung or colon tumor cells. (It is noted that claims are included that recite the activity of stimulation of uptake of glucose or FFA). Applicants have argued that this is a legitimate functional limitation, and that a functional limitation is an attempt to define something by what it does, rather than by what it is. However, the characteristic of being encoded by a gene which is amplified in certain tissues, is not a description of what the protein does. Furthermore, it is maintained that the recitation of the polypeptide variants having as little as 80-85% Homology to the disclosed protein, and having a particular function, does not adequately provide guidance for one of ordinary skill in the art to discover which of the proteins encompassed has the claimed activity. Therefore, the rejection is maintained.

Claims 28-32 and 39-47 are rejected under 35 U.S.C. 1 12, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

This rejection is maintained essentially for the reasons set forth in the previous Office action, mailed 4/10/07.

Applicants have argued that they maintain the position set forth in the previous responses and the Appeal Brief. For the reasons set forth in the previous Office action, the rejection is maintained.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

Claims 28-35, 38, 41-45 are rejected under 35 U.S.C. 102(a) as being anticipated by Youseff et al. (Anticancer Research, 19:2843-2852 (1999)).

Youseff et al. disclose an isolated polypeptide having 100% homology to the protein whose sequence is shown in SEQ ID NO:194 (see attached alignment).

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Nancy T. Vogel whose telephone number is (571) 272-0780. The examiner can normally be reached on 7:00 - 3:30, Monday - Friday.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Joseph Woitach can be reached on (571) 272-0739. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

NV
12/20/07

NANCY VOGEL
PRIMARY EXAMINER

RESULT 1
KLKC_HUMAN
ID   KLKC_HUMAN   STANDARD;   PRT;   248 AA.
AC   Q9UKR0; Q9UKR1;
DT   16-OCT-2001 (Rel. 40, Created)
DT   16-OCT-2001 (Rel. 40, Last sequence update)
DT   16-OCT-2001 (Rel. 40, Last annotation update)
DE   Kallikrein 12 precursor (EC 3.4.21.-) (Kallikrein-like protein 5)
DE   (KLK-L5).
GN   KLK12 OR KLKL5.
OS   Homo sapiens (Human).
OC   Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC   Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
OX   NCBI_TaxID=9606;
RN   [1]
RP   SEQUENCE FROM N.A. (ISOFORM 1).
RX   MEDLINE=20118156; PubMed=10652563;
RA   Yousef G.M., Luo L.-Y., Diamandis E.P.;
RT   "Identification of novel human kallikrein-like genes on chromosome
RT   19q13.3-q13.4.";
RL   Anticancer Res. 19:2843-2852(1999).
RN   [2]
RP   SEQUENCE FROM N.A. (ISOFORMS 1 AND 2).
RA   Yousef G.M., Magklara A., Scorilas A., Diamandis E.P.;
RT   "Cloning of new alternatively spliced forms of the kallikrein-like
RT   gene 5 (KLK-L5).";
RL   Submitted (NOV-1999) to the EMBL/GenBank/DDBJ databases.
RN   [3]
RP   SEQUENCE FROM N.A. (ISOFORM 1).
RX   MEDLINE=20510030; PubMed=11054574;
RA   Gan L., Lee I., Smith R., Argonza-Barrett R., Lei H., McCuaig J.,
RA   Moss P., Paeper B., Wang K.;
RT   "Sequencing and expression analysis of the serine protease gene
RT   cluster located in chromosome 19q13 region.";
RL   Gene 257:119-130(2000).
RN   [4]

RP   SEQUENCE FROM N.A. (ISOFORM 2).
RA   Lamerdin J.E., McCready P.M., Skowronski E., Viswanathan V.,
RA   Burkhart-Schultz K., Gordon L., Dias J., Ramirez M.,
Stilwagen S.,
RA   Phan H., Velasco N., Do L., Regala W., Terry A., Brower A.,
Garnes J.,
RA   Danganan L., Erler A., Christensen M., Georgescu A., Avila
J., Liu S.,
RA   Andreise T., Trankheim M., Attix C., Amico-Keller G.,
Coefield J.,
RA   Duarte S., Lucas S., Bruce R., Thomas P., Quan G., Kronmiller
B.,
RA   Arellano A., Sanders C., Ow D., Nolan M., Trong S., Kobayashi
A.,
RA   Olsen A.S., Carrano A.V.;
RT   "Sequence analysis of chromosome 19q13.4.";
RL   Submitted (OCT-2000) to the EMBL/GenBank/DDBJ databases.
CC   -!- SUBCELLULAR LOCATION: Secreted (Probable).
CC   -!- ALTERNATIVE PRODUCTS:
CC   Event=Alternative splicing; Named isoforms=2;
CC   Name=1;
CC   IsoId=Q9UKR0-1; Sequence=Displayed;
CC   Name=2;
CC   IsoId=Q9UKR0-2; Sequence=VSP_005403;
CC   -!- SIMILARITY: Belongs to peptidase family S1. Kallikrein
subfamily.
CC

CC

DR   EMBL; AF135025; AAD26426.2; -.

```
DR   EMBL; AF135025; AAF06065.1; -.
DR   EMBL; AF243527; AAG33365.1; -.
DR   EMBL; AC011473; AAG23258.1; -.
DR   HSSP; P00763; 1DPO.
DR   MEROPS; S01.020; -.
DR   Genew; HGNC:6360; KLK12.
DR   MIM; 605539; -.
DR   GO; GO:0005576; C:extracellular; NAS.
DR   GO; GO:0004252; F:serine-type endopeptidase activity; NAS.
DR   GO; GO:0006508; P:proteolysis and peptidolysis; NAS.
DR   InterPro; IPR009003; Cys_Ser_trypsin.
DR   InterPro; IPR001254; Peptidase_S1.
DR   InterPro; IPR001314; Peptidase_S1A.
DR   Pfam; PF00089; trypsin; 1.
DR   PRINTS; PR00722; CHYMOTRYPSIN.
DR   SMART; SM00020; Tryp_SPc; 1.
DR   PROSITE; PS50240; TRYPSIN_DOM; 1.
DR   PROSITE; PS00134; TRYPSIN_HIS; 1.
DR   PROSITE; PS00135; TRYPSIN_SER; 1.
KW   Hydrolase; Serine protease; Glycoprotein; Signal;
KW   Alternative splicing.
FT   SIGNAL        1     17       POTENTIAL.
FT   CHAIN        18    248       KALLIKREIN 12.
FT   ACT_SITE     62     62       CHARGE RELAY SYSTEM (BY
SIMILARITY).
FT   ACT_SITE    108    108       CHARGE RELAY SYSTEM (BY
SIMILARITY).
FT   ACT_SITE    200    200       CHARGE RELAY SYSTEM (BY
SIMILARITY).
FT   DISULFID     28    161       BY SIMILARITY.
FT   DISULFID     47     63       BY SIMILARITY.
FT   DISULFID    133    235       BY SIMILARITY.
FT   DISULFID    140    206       BY SIMILARITY.
FT   DISULFID    172    186       BY SIMILARITY.
FT   DISULFID    196    222       BY SIMILARITY.
FT   CARBOHYD     24     24       N-LINKED (GLCNAC. . .)
(POTENTIAL).
FT   CARBOHYD    163    163       N-LINKED (GLCNAC. . .)
(POTENTIAL).
FT   VARSPLIC    236    248       KYVDWIRMIMRNN ->
NSTLVGLGTSWNFNSCQPF (in
FT                                isoform 2).
FT                                /FTId=VSP_005403.
SQ   SEQUENCE   248 AA;  26733 MW;  BB473E98F8BAF703 CRC64;

  Query Match              100.0%;  Score 248;  DB 1;  Length 248;
```

Best Local Similarity 100.0%; Pred. No. 7.9e-255;
Matches 248; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

```
Qy          1 MGLSIFLLLCVLGLSQAATPKIFNGTECGRNSQPWQVGLFEGTSLRCGGVLIDHRWVLTA 60

Db          1 MGLSIFLLLCVLGLSQAATPKIFNGTECGRNSQPWQVGLFEGTSLRCGGVLIDHRWVLTA 60

Qy         61 AHCSGSRYWVRLGEHSLSQLDWTEQIRHSGFSVTHPGYLGASTSHEHDLRLLRLRLPVRV 120

Db         61 AHCSGSRYWVRLGEHSLSQLDWTEQIRHSGFSVTHPGYLGASTSHEHDLRLLRLRLPVRV 120

Qy        121 TSSVQPLPLPNDCATAGTECHVSGWGITNHPRNPFPDLLQCLNLSIVSHATCHGVYPGRI 180

Db        121 TSSVQPLPLPNDCATAGTECHVSGWGITNHPRNPFPDLLQCLNLSIVSHATCHGVYPGRI 180

Qy        181 TSNMVCAGGVPGQDACQGDSGGPLVCGGVLQGLVSWGSVGPCGQDGIPGVYTYICKYVDW 240

Db        181 TSNMVCAGGVPGQDACQGDSGGPLVCGGVLQGLVSWGSVGPCGQDGIPGVYTYICKYVDW 240

Qy        241 IRMIMRNN 248

Db        241 IRMIMRNN 248
```

RESULT 2
CFAD_HUMAN
ID CFAD_HUMAN STANDARD; PRT; 253 AA.
AC P00746;
DT 21-JUL-1986 (Rel. 01, Created)
DT 15-DEC-1998 (Rel. 37, Last sequence update)
DT 15-MAR-2004 (Rel. 43, Last annotation update)
DE Complement factor D precursor (EC 3.4.21.46) (C3 convertase activator)